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REFERENCES

- Africa Fighting Malaria. 2006. Africa Fighting Malaria Responds To Berkeley University Study Into DDT And Neurodevelopment In Children. MedicalNewsToday.com 18 July. Available: <http://www.medicalnewstoday.com/articles/47470.php> [accessed 11 March 2010].
- Attaran A, Roberts DR, Curtis CF, Kilama WL. 2000. Balancing risks on the backs of the poor. *Nat Med* 6:729–731.
- Bate R. 2009. Comment: UN disarms Weapon of Malerial Destruction. Joy Online (Ghana), 29 May. Available: <http://news.mjjoyonline.com/features/200905/30279.asp> [accessed 11 March 2010].
- Bate R, De Lorenzo M. 2007. Reconsider DDT against Malaria. *New Times (Rwanda)* 10 January. Available: <http://www.aei.org/article/25431> [accessed 11 March 2010].
- Roberts DR. 2001. DDT risk assessments [Letter]. *Environ Health Perspect* 109:A302–A303.
- Roberts DR. 2007a. A New Home for DDT. *New York Times*, August 20. Available: <http://www.nytimes.com/2007/08/20/opinion/20roberts.html> [accessed 11 March 2010].
- Roberts DR. 2007b. Preventing malaria in endemic areas [Editorial]. *BMJ* 335:1001–1002; doi:10.1136/bmj.39370.673785.BE [Online 24 October 2007].
- Roberts D, Curtis C, Tren R, Sharp B, Shiff C, Bate R. 2004. Malaria control and public health [Letter]. *Emerg Infect Dis* 10:1170–1171. Available: http://www.cdc.gov/ncidod/EID/vol10no6/03-0787_03-1116.htm [accessed 11 March 2010].
- Roberts DR, Manguin S, Mouchet J. 2000. DDT house spraying and re-emerging malaria. *Lancet* 356:330–332.
- Schapiro A. 2006. DDT: a polluted debate in malaria control [Letter]. *Lancet* 368:2111–2113; doi:10.1016/S0140-6736(06)69812-7 [Online 8 December 2006].
- Tren R. 2002. Africa needs DDT [Letter]. *New York Times*, 28 December. Available: <http://www.nytimes.com/2002/12/28/opinion/1-africa-needs-ddt-590258.html> [accessed 11 March 2010].
- Tren R. 2009. Insecticides for public health. *CMAJ* 180:784; doi:10.1503/cmaj.090358.
- Tren R, Roberts D. 2010. DDT and malaria prevention [Letter]. *Environ Health Perspect* 118:A14–A15; doi:10.1289/ehp.0901276 [Online 1 January 2010].
- van den Berg H. 2009. Global status of DDT and its alternatives for use in vector control to prevent disease. *Environ Health Perspect* 117:1656–1663; doi:10.1289/ehp.0900785 [Online 29 May 2009].

DDT in Malaria Control: Roberts and Tren Respond

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Herren and Mbogo's critique of our response (Tren and Roberts 2010) to van den Berg (2009) is lacking in substance. In their letter, they attack our work by characterizing our advocacy for using DDT to control malaria as a distraction from larger malaria control issues. These authors apparently discount the fact that some African countries are presently making highly effective use of DDT to reduce both malaria deaths and malaria infections. Countries that use DDT benefit from its spatial repellent action that stops mosquitoes from entering houses and transmitting disease, and no alternative insecticide does this (Roberts and

Tren 2010). In addition, Herren and Mbogo apparently do not understand that our advocacy is consistent with that exhibited by the malaria control community, with hundreds signing a petition to prevent DDT elimination through Stockholm Convention negotiations. If DDT had been eliminated, countries presently using DDT would have been deprived of its benefits for protecting health and saving lives. Herren and Mbogo claim that our response to van den Berg's commentary (van den Berg 2009) was fixated on DDT, in lieu of addressing the larger issues of what should be done to control malaria. In our letter (Roberts and Tren 2010), we addressed what we considered to be an attack on DDT use. How could we have responded without addressing the issues in van den Berg's commentary?

Herren and Mbogo mischaracterize our position vis-à-vis DDT and alternative insecticides by asserting that we are reducing the malaria control debate to a simplistic equation of malaria or DDT. In fact, we have a public record of supporting the use of insecticide-treated nets and the use of alternative insecticides for malaria control. However, we have repeatedly emphasized that, for obvious reasons, insecticide-treated nets are not the only solution for malaria control. In fact, we object to a theme of nets and nets alone as much as we would object to a theme of DDT and DDT alone. Basically, there is no single-solution approach to malaria control. All tools are needed—not just those that are currently in vogue.

Herren and Mbogo state that they are fully aware that malaria is a worse outcome than possible health effects of DDT. We agree with them and appreciate their willingness to admit this, because their admission opposes published speculations that DDT might be causing more harm than good (Chen and Rogan 2003).

Herren and Mbogo conclude that we “do more to fuel those ‘interminable debates’ [DDT or no DDT for malaria control] than to meaningfully inform decisions that will save people's lives.” It seems that these authors ignore the fundamental fact that we do not elaborate on alternative approaches to malaria control because the alternatives are not presently under threat of elimination. The alternatives are being used and should continue to be used, but the future is far less certain for DDT. Advocacy saved DDT from being eliminated during the original negotiations for the Stockholm Convention, and lives are being saved and diseases prevented as a consequence. The idea that the threat is over and that DDT is now available to those countries making effective use of it is wrong. The Stockholm Convention Secretariat is now planning to stop all production of DDT in 2017 and eliminate it entirely from use in malaria control programs in 2020 (UN Environment Program 2010).

The Stockholm Convention Secretariat plans to prevent future uses of DDT, even though there is no cost-effective replacement for DDT. Given these circumstances, Herren and Mbogo should expect the interminable debates to become even more polemic in the future.

As for the big issues of what should be done to control malaria, our position is clear: Decisions should be based on scientific evidence of what actually works, on local circumstances, and on what proves to be the most cost-effective in terms of reducing disease and preventing human deaths.

R.T. runs a policy and advocacy group, Africa Fighting Malaria, and both R.T. and D.R. serve on the board of Africa Fighting Malaria. The organization has offices in South Africa and the United States and conducts critical analysis of malaria control programs and funding agencies and strive to build more transparent, accountable, and effective malaria control programs. Africa Fighting Malaria has worked to defend the decisions of malaria control programs to use DDT and to argue for a sound, scientific assessment of the chemical. The organization has a policy of not accepting funds from the insecticides industry and has never received any donations from this sector.

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REFERENCES

- Chen A, Rogan WJ. 2003. Nonmalaria infant deaths and DDT use for malaria control. *Emerg Infect Dis* 9:960–964.
- Roberts DR, Tren R. 2010. The Excellent Powder, DDT's Political and Scientific History. Indianapolis, IN:DogEar Publishing.
- Tren R, Roberts D. 2010. DDT and malaria prevention [Letter]. *Environ Health Perspect* 118:A14–A15; doi:10.1289/ehp.0901276 [Online 1 January 2010].
- UN Environment Program. 2010. Stockholm Convention on Persistent Organic Pollutants (POPs). The Race to Eliminate DDT. Available: <http://chm.pops.int/Programmes/DDT/ActionPlanandProjects/tabid/130/language/en-US/Default.aspx> [accessed 27 April 2010].
- van den Berg H. 2009. Global status of DDT and its alternatives for use in vector control to prevent disease. *Environ Health Perspect* 117:1656–1663; doi:10.1289/ehp.0900785 [Online 29 May 2009].

Traffic-Related Air Pollution and Childhood Asthma

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We congratulate Clark et al. (2010) for their interesting article concerning traffic-related air pollution and asthma in children. They examined early-life (*in utero* and during the first year of life) exposure to traffic-related air pollution in a large population-based study

(a nested case-control study including nearly 3,500 children). The authors found an association between elevated early-life exposure to traffic-related air pollution and a higher risk of asthma in preschool-aged children. However, Clark et al. (2010) address aspects, raise questions, and give results that deserve further comment—both concerning specific items and general “structural” criteria used in epidemiological studies on adverse health effects from air pollution.

Recent reports have suggested that individual susceptibility could play a previously unsuspected role in the occurrence of diseases (Cetta et al. 2009a), perhaps a role greater than that of the intrinsic toxicity of pollutants (Cetta et al. 2009b). This could explain, at least in part, why it is so difficult to determine a precise threshold concentration that is harmful or safe for each individual (Cetta et al. 2007). But this is just one side of the question.

The main question is that, in the absence of adequate and specific markers of exposure, effect and susceptibility, the linear dose and effect model, and the concomitant pollutant concentration and disease occurrence relationship cannot explain the complexity of the phenomenon of host-particle interactions. In particular, initial cell alterations (e.g., oxidative stress, DNA adduct formation) rarely turn into permanent tissue damage and evident disease because of host repair and defence mechanisms.

In their article Clark et al. (2010) noted another important aspect that should be considered when comparing pollutant concentrations with the burden of deleterious effects, both at the individual and population levels: acute effects of peak concentrations of pollutants that lead to acute admission to hospital and the chronic damage that causes long-term effects. In fact, we should consider both of these as separate entities. However, we also should consider the effect of air pollution

on newborns, which greatly depends on individual susceptibility—either congenital or acquired. This could play a major role in future outcomes and shed new light on the peculiar pathophysiological mechanisms of most pollution-related diseases. Clark et al. (2010) correctly outlined the asynchronism and the delay (0–4 years) between the initial pathogenetic exposure to pollutants (*in utero* or during the first year of life) and the occurrence and detectability of clinically relevant asthma. This further adds to the complexity of host-particle interactions.

There are three issues that should be taken into account in developmental epidemiology studies such as that by Clark et al. (2010). First, epidemiological studies that concomitantly evaluate pollutant concentration and detectable diseases or hospital admissions usually neglect the perinatal damage in fetuses and newborns, which is not immediately detectable but is a delayed manifestation.

Second, perinatal damage from air pollution deserves further attention and detailed analysis because it includes fetal malformations, birth defects, and developmental alterations of newborns. Injury from perinatal air pollution exposure could also be responsible for the increased proportion of unsusceptible individuals who, because of their exposure to pollutants during the susceptibility window and because of epigenetic alterations due to environmental factors, will become susceptible. Epigenetic alterations could also transfer this susceptibility to future generations, leading to individuals developing not only asthma at 4 years of age but also respiratory, cardiovascular or systemic diseases ≥ 20 years later.

Third, clinical and pathophysiological details are not “details” but basic issues and questions—still unsolved—that should be primary goals for future research. They are critical to improving design of epidemiologic

studies and to selecting appropriate models, which should also include biological and pathophysiological parameters and variables because they significantly affect clinical outcomes.

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REFERENCES

- Cetta F, Dharmo A, Azzarà A, Moltoni L. 2009a. The role of inherited predisposition and environmental factors in the occurrence of multiple different solid tumors in the same individual. In: *Multiple Primary Malignancies* (Renda A, ed). Milan:Springer Verlag, 157–178.
- Cetta F, Dharmo A, Moltoni L, Bolzacchini E. 2009b. Adverse health effects from combustion derived nanoparticles. The relative role of intrinsic particle toxicity and host response [Letter]. *Environ Health Perspect* 117:A190.
- Cetta F, Dharmo A, Schiraldi G, Camatini M. 2007. Particulate matter, science and European Union policy [Letter]. *Eur Respir J* 30:805–806.
- Clark NA, Demers PA, Catherine JK, Koehoorn M, Lencar C, Tamburic L, Brauer M. 2010. Effect of early life exposure to air pollution on development of childhood asthma. *Environ Health Perspect* 118:284–290.

Editor's note: In accordance with journal policy, Clark et al. were asked whether they wanted to respond to this letter, but they chose not to do so.

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